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Does a pint a day affect your child's pay? The effect of prenatal alcohol exposure on adult outcomes

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Does a pint a day affect your child's pay? The effect of prenatal alcohol exposure on adult outcomes

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cemmap working paper CWP22/08

Does a pint a day affect your child's pay?

The effect of prenatal alcohol exposure on adult outcomes

by

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August 11th, 2008

Abstract

This paper utilizes a Swedish alcohol policy experiment conducted in the late 1960s to identify the impact of prenatal alcohol exposure on educational attainments and labor market outcomes. The experiment started in November 1967 and was prematurely discontinued in July 1968 due to a sharp increase in alcohol consumption in the experimental regions, particularly among youths. Using a difference-in-difference-in-differences estimation strategy we find that around the age of 30 the cohort in utero during the experiment has substantially reduced educational attainments, lower earnings and higher welfare dependency rates compared to the surrounding cohorts. The results indicate that investments in early-life health have far-reaching effects on economic outcomes in later life.

Keywords: Infant health, education, earnings, alcohol policy

JEL-codes: I12, I18, J24

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1 Introduction

Today, prenatal exposure to alcohol is regarded as one of the main preventable causes of mental retardation (Abel and Sokol, 1987; West and Blake, 2005).¹ Since the early 1970s, medical and epidemiological research has collected a considerable body of evidence supporting a negative association between prenatal alcohol exposure and children's health. The range of damage includes mild and subtle changes, such as slight learning difficulties or physical abnormality, through full-blown Fetal Alcohol Syndrome (FAS) including severe learning disabilities, growth deficiencies, abnormal facial features, and central nervous system disorders. While the short run effects of maternal consumption of alcohol during pregnancy on child health are relatively well covered, the extent of the long-run consequences is not fully known. However, estimating the causal effect of prenatal exposure to alcohol on child outcomes, particularly in the long run, is complex. In particular, unobserved characteristics directly related both to the child's outcomes and maternal alcohol consumption (e.g. poverty or maternal mental health) makes the interpretation of nonexperimental estimates difficult.²

This paper attempts to isolate the causal relationship between prenatal alcohol exposure and adult outcomes. To do this we investigate the impact of a Swedish alcohol policy experiment on the educational and labor market outcomes of the children in utero

¹ Still in the US up to 50 percent of the childbearing age women drink and 16 percent of them continue drinking during pregnancy (CDC, 2002). Göransson et al. (2003) surveyed pregnant women in Stockholm, Sweden regarding their consumption of alcohol. They found that 46 percent reported a binge drinking (more than 5 standard drinks on a single occasion) episode once per month or more often in the year prior to becoming pregnant. During pregnancy 30 % reported regular alcohol use. In a Danish study, 57 % of the pregnant women without previous children reported at least one binge drinking episode during the first half of the pregnancy (Kesmodel et al., 2003). See WHO (2004) for international consumption levels.

during the experiment. During the experiment, alcohol availability in two treatment regions (jointly containing 12 % of the Swedish population) increased sharply as regular grocery stores were allowed to market strong beer³. Prior to and after the experiment, off-premises sales of strong beer, wine and spirits were only allowed in the state-owned alcohol retail monopoly stores (Systembolaget). The experiment was planned to run from November 1967 until the end of 1968 but was discontinued prematurely due to alarming reports of a sharp increase in alcohol consumption in the treatment regions, particularly among youths (SOU 1971:77). *Figure 1* depicts the trend in strong beer sales for the treatment regions and the country as a whole from 1962 through 1972. During the first six months of 1968, strong beer consumption per capita increased ten-fold in the treatment regions as compared to the year prior to the experiment.

The temporary and exogenous increase in alcohol availability during the experiment provides us with a unique opportunity to solve many of the identification problems present in previous work. Firstly, due to its sharp restriction in time, the experiment allows for a comparison of the adult outcomes of the cohort of children born in the experimental regions who were in utero during the experiment with the outcomes for the surrounding “unexposed” cohorts. Secondly, the spatial restriction allows for a simultaneous comparison with the outcomes for children belonging to the same cohort but who were born in the control regions. This feature reduces the problem of general time effects confounding the estimates of the relationship of interest. Thirdly, we capitalize on the fact that the experiment increased alcohol availability relatively more

² Additionally, eliciting correct information on maternal alcohol use during pregnancy is complicated by desirability and recall biases.

³ Strong beer is restricted to a maximum alcohol content of 4.48 % by weight.

for young people under the age of 21 compared to older individuals. This is due to a minimum alcohol purchasing-age law prohibiting youths below age 21 from buying strong beer (and other spirits) at the Systembolaget stores prior to and after the experiment.⁴ During the experiment, the age limit was 16 in grocery stores.⁵ By comparing the outcomes of the children born by mothers below the age of 21 at the date of birth with the outcomes for children born by older mothers, any differing trends in outcomes between children born in treatment and control counties are taken into account. Finally, by focusing on the outcomes of the cohort in utero during the experiment but conceived *before* it started we are able to mitigate the concern that the experiment also might have altered the composition of births and thereby indirectly the child's outcomes.⁶

Using administrative data on all children born between 1964 and 1972, we find that the sharp increase in alcohol consumption during the experiment has had a substantial impact on the outcomes of those still in utero during the experiment. In particular, we find that the children with the longest prenatal exposure to the experiment (between 5 and 8.5 months in utero) who were born by mothers under the age of 21 at delivery have on average fewer years of schooling and lower high school and college graduation rates. They are less likely to be employed, have lower earnings and a higher welfare dependency rate compared to the surrounding cohorts. The results also suggest that males are more vulnerable to adverse conditions in utero (due to alcohol exposure), as

⁴ Several previous studies on consumption by young people have found responsiveness to policies pertaining to availability, such as the minimum legal drinking age (MLDA) laws, see e.g. Moore and Cook (1995).

⁵ See SFS (1967:213) and SFS (1961:159) for the rules in effect during the experiment.

the effects on late-life outcomes are in general more pronounced among males. Similarly, previous work has related a reduced sex-ratio at birth to adverse maternal conditions during pregnancy.⁷ In line with these results we find that the proportion of males in the most exposed cohorts is significantly reduced.

This is the first study focusing on the long term economic impact of prenatal exposure to alcohol. We also contribute to an emerging literature examining the importance of early-life health conditions for subsequent outcomes.⁸ With a few exceptions⁹, the previous work has focused on infant health. Our study distinguishes itself from most of the previous work on early-life conditions and adult outcomes by providing relatively clear suggestions for policy tools that legislators promoting equal opportunities in health and education may use. Furthermore, the results suggest that investment in early-life health may not only be more humane compared to postnatal investment in terms of health outcomes, but potentially also a more effective way of increasing human capital accumulation in comparison with investments later in life (Almond, 2006).¹⁰

⁶ See Kaestner and Joyce (2001) for evidence of the effects of alcohol use on the probability of unwanted pregnancies. In a recent study Watson and Fertig (2008) find evidence suggesting that MLDA laws affects infant health adversely mainly through its effect on composition of births.

⁷ C.f. Triver and Willard (1973), Wells (2000), Norberg (2004), Almond and Edlund (2007).

⁸ c.f. Currie and Hyson (1999), Currie (2008) and the references there in.

⁹ For example, Van den Berg, Lindeboom and Portrait (2006) investigate the impact of early life economic conditions on mortality later in life; Case, Fertig and Paxson (2005) quantify the lasting effects of childhood health and economic circumstances on adult health and earnings; Banerjee et al. (2007) find that economic conditions during childhood decreases stature among males but not life expectancy for females. Utilizing twin data, Black et al. (2007) shows that low birth weight (a common proxy for adverse conditions in utero) is strongly negatively correlated with cognitive ability and stature at age 18-20 as well as subsequent labor market outcomes. Almond (2006), and Almond and Mazumder (2005) investigate the impact of the Spanish influenza pandemic on subsequent socio-economic and health outcomes respectively of those in utero during the peak of the epidemic. Almond, Edlund and Palme (2007) study the impact of the Chernobyl accident on Swedish children exposed to the fallout while still in utero and finds significant negative effects on educational attainments.

¹⁰ See Heckman (2007) for a life-cycle investment framework for human capabilities formation.

The remainder of the paper is structured as follows. Section 2 provides an overview of previous work on the consequences and mechanisms of prenatal alcohol exposure on child development and also the implementation and results of the policy experiment. Section 3 describes the data and the empirical strategy. Section 4 presents the results and robustness checks and section 5 concludes.

2 Prenatal alcohol exposure and the policy experiment

2.1 Consequences of prenatal alcohol exposure

While the medical professions beliefs regarding the impermeability of the placenta were shattered in the early 1960s in connection with the Thalidomide tragedy (see e.g. Dally, 1998), the first scientific support on a negative association between heavy maternal alcohol consumption during pregnancy and children's health did not emerge until 1968 in work by Lemoine et al. (1968) in France. Jones and Smith (1973) subsequently published similar findings and coined the Fetal Alcohol Syndrome (FAS).¹¹ In addition to confirmed maternal alcohol consumption during pregnancy, the FAS diagnosis criteria require the following conditions in infancy: growth deficiency, facial anomalies and neurological abnormalities. Other effects associated with prenatal alcohol exposure are increased risk of miscarriage and low birth weight. Many children that are not obviously physically affected, or do not show any easily defined behavioural problems may still suffer from alcohol-induced central nervous system deficits. Streissguth et al. (1991) demonstrated that there is a predictable long-term progression of disorders into

adulthood resulting from prenatal exposure to alcohol. They show that, among other things, poor judgment, distractibility, difficulty in perceiving social cues and low IQ levels, were common among individuals exposed to alcohol in utero.¹² The evidence on the consequences of medium and lower levels of alcohol consumption during pregnancy on birth outcomes is, however, less conclusive.¹³ Yet, no consensus has been reached on any threshold level, either in terms of the amount or incidence of alcohol consumption during pregnancy with regards to the more subtle effects on health.¹⁴

West et al. (1994) and Goodlet and Horn (2001) summarize the vast medical literature focusing on the particular biological mechanisms that play in the casual link between alcohol exposure and fetal development. Briefly, alcohol may affect the developing fetus directly as it readily crosses the placenta and passes to the fetal cells, but also indirectly by reducing the supply of oxygen and nourishment. In addition, the dose and pattern of alcohol use seem to be important in determining the severity of the damage. Animal experiments have suggested that a small dose consumed in a massed “binge drink” manner is more damaging than a larger but more spaced dose (Bonthius and West, 1990).¹⁵ Furthermore, the detrimental effect of alcohol on fetal development is difficult to isolate to any specific timing of exposure during gestation, although the types of damage may vary between trimesters. While the central nervous system is susceptible to damage during all three trimesters, animal studies suggest that the third

¹¹ Olegård et al. (1979) is the first to study using Swedish data to estimate the effects of prenatal alcohol exposure on child outcomes. They find that alcohol exposure is related to an increased level of behavioral problems in childhood.

¹² The set up and findings from this and other studies on the same cohort of children followed from birth to the age of 25 and born in Seattle in 1974/1975 is summarized in Streissguth (2007). In common to the present study the information on maternal alcohol consumption was elicited when very little was known about the risks associated with alcohol use during pregnancy.

¹³ See e.g. Rusell (1991) and Henderson et al. (2007) for reviews of this literature.

¹⁴ See e.g. CDC (2004).

trimester is a specially sensitive period for the brain (Marcussen et al., 1994). On the other hand, this pattern is less clear-cut for behavioural outcomes among human subject.¹⁶ In addition to direct effects on the central nervous system and brain development, prenatal alcohol exposure may also alter the development and functioning of the immune system, leading to a higher susceptibility to infections (Zhang et al., 2005). Damage inflicted on other organs and extremities mainly seems to occur as a result of exposure in the first trimester. Hence, prenatal alcohol exposure may reduce the health stock through several different paths.

2.2 Swedish alcohol policy and the strong beer experiment

Alcohol sales in Sweden are strictly regulated by means of an off-premises retail monopoly (Systembolaget). The only alcoholic beverages permitted in regular grocery stores are those containing less than 3.5 % alcohol by volume (~2.8 % by weight). The current form of the alcohol retail system has been in effect since 1955. Since then, the consumption pattern has changed radically. Sweden traditionally belonged to the “spirit-drinking” countries, but during the last 50 years the consumption of spirits has declined substantially and has gradually been replaced by wine and beer products (Leifmann, 2001). The dominant alcoholic beverage today is the strong beer that accounts for 29 % of total alcohol consumption (SNIPH, 2005). One of the contributory factors in this changing pattern is active measures taken to encourage the substitution of consumption from spirits to beverages with a lower alcohol content.¹⁷

¹⁵ This is consistent with the results from Streissguth et al. (1990, 1994) which found a binge drinking consumption pattern to be the best predictor of academic achievements.

¹⁶ c.f. Coles (1994) for a discussion of the difficulties of identifying critical periods of alcohol exposure on offspring outcomes in humans.

¹⁷ See Room (2002) for a comprehensive review of Swedish and Nordic alcohol policies after 1950.

The experiment with free sales of strong beer (maximum alcohol contents of 5.6 % by volume, i.e. ~4.48 % by weight), running from November 1967 through July 1968 in the *Göteborgs-och Bohuslän* and *Värmland* counties was one example of a policy of this nature.¹⁸ During the experiment, off-premises sales of strong beer were allowed in regular grocery stores as compared to only in the Systembolaget stores prior to and after the experiment.¹⁹ The regulations for wholesale trading in strong beer also changed. Anyone entitled to sell or serve beer was allowed to buy strong beer directly from a Swedish brewery or, in the case of imported beer, through a wholesaler. The aim of the experiment was that the wholesaling of strong beer was also to be carried out under similar conditions as those that would exist with free sales. As a result, wholesalers were able to order goods directly from foreign breweries.²⁰

The original intention was to continue the experiment until the end of 1968, but soon after it was introduced reports of a sharp increase in alcohol consumption in the experimental counties, especially among youths was received. This caused the implementing authority, the Alcohol Policy Commission (APU), to propose an interruption, and in the middle of July 1968 the experiment was discontinued prematurely.

The consumption of strong beer increased dramatically in the experimental regions during the experiment. In the first half of 1968 consumption increased from the 1967

¹⁸ The setup and results of the experiment are described in detail in the APU report from the experiment (SOU 1971:77), upon which this section draws. In the report no motivation is given as to why the two counties were selected from the pool of 25 counties.

¹⁹ At the end of 1968, 1 530 retail outlets were licensed for sales of beer (during the experiment also strong beer) in *Göteborg och Bohuslän* county as compared to the 26 Systembolaget stores in operation prior to and after the experiment.

²⁰ During the experiment all wholesalers were however obligated to report the amount of strong beer shipped to retailers.

level of 1.2 (0.32) million litres (gallons) to 10.5 (2.77) million litres (gallons) in Göteborgs- och Bohuslän. In Värmland the increase was even more drastic. In the first six months of 1967 0.2 (0.05) million litres (gallons) were sold compared to 3.0 (0.79) million litres (gallons) during the same months in 1968. Summarized over both regions, consumption increased almost ten-fold. Per capita, the consumption of strong beer increased from 1.8 (0.48) litres (gallons) during the first six months of 1967 to 15.3 (4.04) litres (gallons) in the same period in 1968 in Göteborgs- och Bohuslän. The corresponding figures for Värmland were 0.7 (0.18) litres (gallons) and 10.6 (2.8) litres (gallons) per capita for the two periods. From *Figure 1* we also see that consumption in the country as a whole rose during the experiment. The main part of this increase is explained by the fact that the two experimental counties constituted a substantial share of the total population (12 % in 1968) and hence had a large impact on the national average. If we exclude the experimental regions, the rest of the country showed an increased consumption of 26 % from the first half of 1967 to the same period in 1968. From *Figure 1* we can also see that before the experiment the trends in consumption of strong beer in the two experimental counties followed the national average reasonably well. During the experiment, consumption boomed and afterwards it fell back again. However, it should be noted that strong beer consumption in the experimental regions remained at an elevated level compared to the pre-experiment period even after the experiment had ended. This indicates that a short-term experiment could have long-term effects on consumption (SOU 1971:77).

The geographical distribution of consumption reveals a clear connection between sales and population density. Per capita consumption was highest in Gothenburg

(684,626 inhabitants) followed by Karlstad (53,208 inhabitants) and Uddevalla (36,480 inhabitants). The reason for this pattern is probably greater availability in urban areas. Another explanation might be that people living in rural areas bought strong beer when visiting the cities. However, it is also likely that some cross-border shopping for beer occurred during the experiment at least by consumers in the neighbouring counties. This suggests that an experiment including the whole country would have generated a smaller increase in consumption per capita. The extent of cross-border shopping is unknown but it seems unlikely that it had any major influence on total sales.²¹

There are excellent opportunities for evaluating the impact of the experiment on substitution between wine, spirits and strong beer. The Systembolaget stores kept exact records of the volumes sold per quarter in each county prior to, during, and after the experiment. Compared to the first half of 1967, there was a decrease in liquor sales in the first half of 1968 in the two experimental regions of ten and of five percent respectively, while the wine sales did not change to any great extent. For the rest of the country, the decline in liquor sales was four percent, while the wine sales increased by eight percent. These figures indicate that the experimental regions differed from the rest of the country by having larger decreases in liquor sales and no increase in wine sales. This suggests that, in the experimental regions, liquor and wine was substituted by strong beer. The changes in liquor and wine sales were, however, rather small and did not compensate for the substantial increases in sales of strong beer.

²¹ The reason is that while availability increased, prices (if anything) increased during the experiment (SOU 1971:77). In the empirical section, however we check whether the experiment generated any spill-over effects on children born in the neighboring counties.

Perhaps a more important question is how the consumption of medium beer²² was influenced. It is highly likely that the increased sales of strong beer lead to a decline in the sales of medium beer, as these products are arguably closer substitutes. Unfortunately, there are no records of the quantity of medium beer sold at the county level. There are however data on aggregate monthly sales. The national consumption of medium beer increased by only 14 % during the first six months of 1968. This should be compared with an increase of 25 % for the first three quarters of 1967 and 35 % during the fourth quarter of 1968. These figures indicate that the experiment led to a reduction in the increase of medium beer sales of 10 percentage points, and that strong beer to some extent replaced medium beer in the experiment regions. During the first six months of 1967, 91 (24) million liters (gallons) of medium beer was sold, which means that the reduction should have been around 10 (2.6) million liters (gallons) overall. This quantity should be compared with the extra 11.8 (3.1) million liters of strong beer consumed in the experimental counties. Based on these calculations, the average increase in the experimental regions in terms of litres of 100 % alcohol has previously been estimated to be not more than five percent (SOU 1971:77). However, potential heterogeneous consumption responses to the increased availability between different sub-populations have not been taken into consideration.

The immediate impact on harms was only assessed in terms of number of persons arrested for drunkenness. These data show no clear effects of the experiment. However, during this period there was a general increase in alcohol consumption and a general decline in the number of persons apprehended for drunkenness. There were also reports

²² Medium beer may contain at maximum 3.6 % alcohol by weight.

suggesting that the police authorities acted on drunkenness in ways which did not show up in the official statistics (SOU 1971:77). Moreover, in the late spring of 1968 the implementing authority, the Alcohol Policy Commission, surveyed the local child welfare commissions (barnavårdsnämnder), the temperance commissions (nykterhetsnämnder), the local education authorities and the police authorities in the experimental counties regarding their experiences of the free sales of strong beer hitherto. The main conclusion of this survey is that there was a negative impact on temperance during the experimental period. The police authorities underscored that the temperance situation had deteriorated particularly among young people. The main nuisances reported were an increased level of disorderly conduct and litter in connection with an immense consumption of strong beer. An increase in drunken driving was also noted. Furthermore, urban areas seem to have been more affected than rural areas (SOU 1971:77).

One explanation of the particularly detrimental effects on temperance among youths is probably that they experienced the largest increase in the availability of alcoholic beverages during the experiment.²³ The age limit in Systembolaget stores was set to 21, and prior to the experiment this was the only place where strong beer could be bought. The minimum purchasing age for beer in regular grocery stores during the experiment was 16, although the application of this law was very weak (SOU 1974:91).

The main problem for estimating the effect of the experiment on consumption is the lack of data on alcohol use among sub-populations in the experimental counties.

²³ For the effects of alcohol availability on consumption patterns in general see e.g. O'Malley and Wagenaar (1991) for US evidence, Carpenter and Eisenberg (2007) for Canadian evidence, and Norstrom and Skog (2005) for Sweden. For studies focusing on young people see e.g. Moore and Cook (1995).

However, we know from a nationwide survey among youths aged 15 through 25 conducted in the summer of 1968, that beer consumption was 44 % higher among youths than in the population as a whole. This suggests that the average increase in consumption among young people likely exceeds the previously estimated average increase of five percent. The survey also reveals that in 1968, 90 percent of the females and 97 percent of the males reported that their alcohol début occurred before turning 21 and that the abstainer rates in these age categories was low²⁴ (SOU 1971:77).

Considering the type of weekend binge drinking pattern common in Sweden²⁵, the reports of a sharp deterioration in temperance among young people and the particularly damaging effects on the fetus of binge drinking, we believe that the long-run outcomes of children exposed to the experiment in utero may have been affected. The following section describes the data and the empirical strategy we employ to identify the prevalence and importance of any such effects.

3 Data and empirical strategy

3.1 Data and sample selection

The main hypothesis we aim to test in this paper is whether the exogenous increase in alcohol consumption during the experiment resulted in adverse adult outcomes for the children in utero at the time. To do this we utilize the LOUISE database assembled by Statistics Sweden covering all individuals in the age range 16-65 living or working in Sweden between 1990 and 2004. The LOUISE data are register-based and, apart from

²⁴ In the highest, middle and lowest social strata 2, 8 and 10 percent of the young women (aged between 17 and 25) reported no alcohol consumption in 1968 (SOU 1971:77).

information on year and month of birth, gender and county of birth, they also contain detailed information on educational attainments, labor market outcomes and welfare payments received during the observation period. Using the so-called “multi generational” register, we have also linked each individual in the data to his/her biological parents.

In the main analysis, we retain all first-born individuals alive in 2000 and born in Sweden between 1964 and 1972. We exclude children born in the 5 counties neighbouring the experimental counties in order to avoid diluting the estimates due to potential spill-over effects from the experiment. As the experiment was conducted at the county level, this paper uses panel data for counties. However, for the reasons discussed above, we also allow for potential differential effects of the experiment on children of young (below the age of 21 at delivery) and older mothers.

We divide the children born in the treatment counties in the selected cohorts into four groups depending on their exposure status: (1) those born prior to the initiation of the experiment, and hence only exposed after birth; (2) those exposed to the experiment in utero but conceived before the experiment started; (3) those exposed to the experiment in utero but conceived during the course of the experiment; and (4) those who were conceived after the end of the experiment and who, as a result, were not exposed either during pregnancy or after birth.²⁶

In our baseline estimations, we focus in particular on children belonging to group (2). The main reason is that we can be fairly certain that the experiment did not affect the

²⁵ The pattern of drinking in Sweden has been characterized by non-daily drinking, irregular binge drinking episodes (e.g. during weekends and at festivities), and the acceptance of drunkenness in public; see e.g. Kühlhorn et al. (1999).

timing of conception for this group of children. This is important, as several studies have found an association between alcohol consumption and risky behaviour among young people (Kaestner and Joyce, 2001; Carpenter, 2005; Grossman and Markowitz, 2005). Indeed Watson and Fertig (2008) find evidence suggesting that Minimum legal drinking age laws affect infant health mainly through its effect on the composition of births. Hence, by focusing on children conceived prior to the experiment started, we effectively avoid attaining biased estimates of the relationship of interest due to indirect effects caused by the experiment (e.g. via an increased frequency of unplanned pregnancies).

To allow for heterogeneous effects of the experiment depending on duration and/or timing of exposure during gestation, we split the children in group (2) into those whose mothers were in the first half of the pregnancy period (months 1-4), and those in the second half (months 5-9) at the start of the experiment. The first group (months 1-4) probably experienced a particularly high risk of being exposed to alcohol due to the experiment. Partly so due to the long duration of exposure in utero, but also because mothers in early pregnancy probably responded more strongly to an increase in alcohol availability compared to mothers in late pregnancy.²⁷ Moreover, a substantial proportion of the early-pregnancy mothers probably did not even realize that they were pregnant for some time during the experiment.²⁸ This may also have increased the probability of changing their consumption pattern due to the increase in availability.

²⁶ Table A 1 in Appendix A presents a schematic overview on the estimated maximum and minimum number of weeks of in utero exposure, as well as the estimated gestational age at the start of the experiment.

²⁷ In the second part of the empirical analysis we also investigate the impact of the experiment on children in late pregnancy and the three other exposure groups.

²⁸ Today, the average pregnancy is recognized 5-6 weeks after conception (see e.g. Floyd et al., 1999). In the late 1960s, recognition on average probably occurred later due to the lack of readily available pregnancy tests.

One should furthermore bear in mind that the awareness of the risks associated with alcohol consumption during pregnancy was very low at the time of the experiment.

3.2 A first look at the data

Table 1 presents descriptive statistics for the adult outcomes of children born in the control and treatment counties for the cohorts in utero prior to, during and after the experiment. All averages are calculated using data aggregated to the county-by-quarter of birth-by-old/young mother-level and weighted by the number of children in each cell. In all there are 1,748 cells including 353,742 children. The first panel of *Table 1* reports the mean of the outcome variables for children born in the treatment counties and the control counties. Columns 1-6 report averages for children born in the experimental counties (columns 1-3) and the control counties (columns 4-6). Columns 7-12 report the corresponding characteristics for children of mothers under the age of 21 at the date of birth. The statistics in *Table 1* are calculated for the cohorts born during the first two quarters of each year. This table also presents the fathers and mothers ages at the date of birth, the fraction of mothers with a high school diploma (measured in 1990), and the average number of children in each cell. From these (few) background characteristics an increasing age trend among mothers may be noted, and also that the number of young mothers decreases over time in both the treatment and the control counties. Looking at the average outcomes, it appears that the children of the young mothers exposed to the experiment (i.e. born in 1968) tend to have a less favorable development in terms of educational and labor market outcomes compared to the other cohorts.

To get a clearer view of the trend in the outcomes of children born around the time of the experiment, *Figure 2* plots average years of schooling completed in 2000 for

children born between 1966:Q1 to 1970:Q4 by mothers under the age of 21 in the treatment and control counties. The average years of schooling of the treatment county children conceived just prior to the experiment (born during the second quarter of 1968) deviate clearly from the pattern displayed by the adjacent cohorts and the control county cohorts.

A similar pattern is found in *Figure 3* in which the comparison group is now children born in the treatment counties, but with mothers older than 20 at the date of birth. There is no visible change in the educational outcome for children with older mothers, but the dip in years of schooling is still apparent for the young mothers' children. The pattern in the two figures is clearly in line with the police reports suggesting that young people's alcohol consumption increased most during the experiment. The timing also corresponds well with the estimated duration of exposure as presented in *Table A 1*.

Investigation of to what extent the effects carry over into labor market outcomes is also of interest. *Figure 4* plots the average earnings²⁹ at age 32 for the children whose mothers were under the age of 21 on delivery in the control and treatment counties. As in the case of education there is a distinct decrease in relative earnings between treatment and control county children that coincides with the timing of the experiment.

The cumulative earnings distribution of men and women born during the second quarter of 1968 is shown in the left hand side of *Figure 5* in order to get a better picture of where this variation stems from. The cumulative earnings distributions suggests that men at the lower end of the distribution seems to have been strongly affected as the

distribution is pushed to the left for the exposed cohort. In contrast, the earnings differences between those born in the control and treatment counties earning above the 50th percentile are relatively small. Under the assumption that in the absence of the experiment the treated children would have ended up at the same position of the distribution, the experiment seems to mainly have affected low-SES children.³⁰ For comparison, the same distributions are shown on the right hands side of **Figure 5** for individuals born one year prior to the experiment. Again, the difference in distribution between the control and treatment county for this cohort is minimal.

Finally, *Figure 6* plots the proportion of males in corresponding cohorts. Clearly the variance is higher in this case; but still there is a distinct drop in the proportion males, coinciding with timing of the experiment and the changes in the other outcomes. Previous studies have found that a reduced sex-ratio at birth is indicative of adverse maternal conditions during pregnancy (see e.g. Trivers and Willard, 1973; Lee et al., 1998; Wells, 2000). We explore this finding in more detail below.

3.3 Estimation strategy

The descriptive analysis above does indeed suggest substantial drops in average outcomes, coinciding with in utero exposure to the experiment. To gauge more formally to what extent this drop is indeed caused by the experiment, we employ a difference-in-difference-in-differences design and estimate the following reduced form model:

²⁹ The data used in the figure have been trimmed so as to omit individuals with yearly earnings below the 1st percentile (SEK 1400) and above the 99th percentile (SEK 563,700).

³⁰ The invariant rank assumption may however be a strong assumption in this context. A survey among young people aged 15-25 conducted in the spring of 1968 revealed a clearly positive correlation between alcohol usage among young women and the father's socio-economic status (see e.g. SOU 1971:77), suggesting that children of more well-off mothers may actually have been those with the highest exposure.

$$\begin{aligned} \text{OUTCOME}_{c,t,mom<21} = & \alpha_0 + \beta_1 \text{TREATED}_{c,t,mom<21} + \eta_c + \delta_t + \phi_{mom<21} \\ & + \gamma_{c,t} + \lambda_{c,mom<21} + \mu_{t,mom<21} + \varepsilon_{c,t,mom<21} \end{aligned} \quad (1)$$

using data aggregated by birth quarter, age of mother (below/above 21) and county of birth.³¹ In equation (1) *OUTCOME* is the outcome of interest (average years of schooling, share of high school graduates, share on welfare, average earnings etc.). *TREATED* is equal to 1 if the child is born by a mother under the age of 21 at delivery in the treatment counties and conceived between July and October 1967, and otherwise 0.³² Thus β_1 is the parameter of interest and it reflects the impact of the experiment on the outcomes of the children in utero at the time in adulthood. δ_t and η_c are period (quarter/year) and county of birth effects respectively. $\phi_{mom<21}$ is a parameter indicating whether the child was born by a mother under the age of 21 at the date of birth. The time (δ_t) and county (η_c) parameters, respectively, control for county and quarter of birth specific effects that affect the outcomes.³³ The $\phi_{mom<21}$ parameter accounts for fixed differences in outcomes between children born by mothers under the age of 21 and those above. The interaction terms $\gamma_{c,t}$, $\lambda_{c,mom<21}$ and $\mu_{t,mom<21}$ account for many other factors that are also related to the outcomes of interest. For example, as seen in *Table 1*,

³¹ The aggregated data is used instead of individual level data as the treatment varies at this level. The aggregate data is preferred in order to avoid problems of within-county correlations in the error term which may otherwise result in seriously underestimated standard errors as Donald and Lang (2007) show. Using raw aggregated data, as is done in this case yields qualitatively similar results as when using the residual aggregation method, and hence adjusting for background characteristics available in the data as suggested by e.g. Bertrand et al. (2004).

³² Hence in the main estimations the “quarter” of birth is defined as Q1=Jan.-March, Q2=April-July, Q3=Aug.-Sept., Q4=Oct-Dec, so as to be in a better position to capture the full effect on those conceived just prior to the experiment.

³³ See Costa and Lahey (2005) and Dobelhammer and Vaupel (2001) for the importance of season of birth effects on adult health.

over the observation period the number of mothers under the age of 21 decreased somewhat and hence the composition of these mothers may have deteriorated in terms of parental ability, for example. The quarter*youngmom effect ($\mu_{t,mom<21}$) account for such compositional changes throughout the observation period. The county*young mom effects ($\lambda_{c,mom<21}$) control for fixed cross county differences in the composition of mothers giving birth to children under the age of 21. The error term $\varepsilon_{c,t,mom<21}$ is assumed to be IID and potentially heteroskedastic.

We estimate the model in equation (1) by OLS. The identifying assumption needed for a consistent estimate of β_1 is rather weak. There can be no change in unobserved factors coinciding with the timing of the experiment that only affect the adult labor market outcomes of children born by mothers under the age of 21 at the date of birth in the experimental regions. While we can never test this assumption directly, in the following section we report a number of robustness checks in addition to the baseline difference-in-difference-in-differences estimates, in order to validate our estimation strategy. All regressions are weighted by the number of children in each cell. The reported standard errors are robust with respect to heteroscedasticity.

4 Results

To preview the results we find that children exposed to the experiment for the longest duration in utero have significantly lower earnings, higher probability of no earnings at all, lower educational attainments and higher welfare dependency rates. Moreover, we find that males seem to be particularly affected by adverse conditions in utero since for most outcomes, the effects of the experiment are more pronounced for males than for

females. The cohort most highly exposed in utero is furthermore significantly more female. We also find that while there is no significant effect on the month of birth of females in the most highly exposed cohorts, there is a negative effect on the month of birth of males. These two findings indicate that those most heavily exposed to the experiment were more likely to be either spontaneously aborted or born prematurely. The results are furthermore robust to a number of specifications changes.

4.1 Main results

This section reports baseline results from regression analysis based on the specification in equation (1). Panel A, B and C of *Table 2* report estimates of β_1 using the average years of schooling, the proportion high school graduates and the proportion with at least 3 years of higher education as the dependent variable, respectively. Columns (1)-(3) in each panel provide the estimates employing the full sample, the male sample, and finally the female sample. Educational attainment is measured in 2000 when the children in the sample were aged between 28 and 36.

As seen in *Table 2*, the impact of the experiment on educational outcomes is substantial. In the full sample, the coefficient suggests that the number of years of schooling is reduced by 0.27 years on average. Among males, this effect is even stronger - males from the cohort in utero during the experiment have on average 0.47 fewer years of schooling, and among females this effect is somewhat weaker (0.10 years), but not statistically distinguishable from zero at any conventional significance level. Turning to the proportion who graduated from high school, it appears that the children in the exposed cohort are about 4 percentage points less likely to have completed high school. Again, this effect is driven by a lower high school completion

rate of 10 percent with respect to the mean among males ($-0.09/0.9$). The proportion of males who has graduated from higher education is also significantly reduced by 3.9 percentage points, and by 2.1 percentage points for females, but imprecisely estimated. The effect on the proportion of males graduating from higher education is even larger than the effects on the high school completion rates, which support the notion that many children who are not obviously affected by prenatal alcohol exposure may still suffer from cognitive deficits. With respect to the mean, exposed males are about 35 percent ($-0.039/0.11$) less likely to have graduated from higher education.

Moving on to the impact on labor market outcomes presented in *Table 3*, we find that males and females in this case are similarly affected. On average the exposed cohort has close to 24 percent lower earnings at the age of 32. Again, males seem to have been somewhat more strongly affected than females. However, the assumption that women's earnings at the age of 32 are an accurate measure of their permanent earnings is questionable. Böhlmark and Lindqvist (2006) estimate of life-cycle biases shows that, in the case of Sweden, the ideal solution would be to use earning after the age of 40 in order to get a decent proxy for permanent earnings.

Panel B in *Table 3* presents the results of a regression using the fraction with zero earnings as the dependent variable. In this case, the experiment increased the risk of having no labor income at all at age 32 for both men and women with around 7 percentage points. The last panel in *Table 3* reveals that the proportion on welfare among the exposed males is 5 percentage points higher in the exposed cohort. The proportion of females on welfare is also higher, but the impact is not statistically different from zero.

To summarize: for education and labor market outcomes the estimated impact of the experiment is considerable. In the case of education, the effects are comparable with the estimates for other types of insults in utero on subsequent educational attainments (see e.g. Almond et al., 2007; Barreca 2007). Moreover, as shown in *Figure 5* the greatest impact is found at the lower end of the earnings distribution. This indicates that the estimated earnings effect largely reflects labor supply rather than wages. Unfortunately we do not have access to data on wages for this sample. Moreover, while the log transformation of the earnings simplifies interpretation, it also emphasizes differences at the lower end of the earnings distribution. Running the same regression on the non-logged earnings (still excluding the zeros) does reduce the point estimate significantly to around 15 percent, which is still a sizeable effect.

4.2 Differential effects by maternal education and earnings

A higher level of parental resources may potentially mitigate some of the negative effects of health shock early in life. Panel A in *Table 4* reports estimates for the sample of children with mothers with a high-school degree and for those who do not separately. The point estimates for education and labor market outcomes tend to be larger for children of mothers without a high school degree and are more precisely estimated. Panel B in *Table 4* presents the results from the same specifications, but for mother with above or below the median income level in 1990. The pattern is similar in this case with smaller estimated effects for higher income-level mothers.

These set of results indicates that parental resources may mitigate the effects of poor health in childhood on outcomes later in life. However, while suggestive these results should be interpreted with care as the standard errors are large in some cases. Moreover,

the highest educational level and earnings of the mother (both measured in 1990), might potentially be endogenous with respect to the health of the child (see e.g. Powers, 2001).

4.3 Further results and robustness checks

The pattern from tables above is clear. The alcohol experiment seems to have resulted in significantly worse adult outcomes for the children in utero during the experiment. Notably, males seem to have been particularly affected. Why then should males be more strongly affected by an increased prenatal exposure to alcohol than females? The results in *Table 4* provide some guidance. The table reports coefficients from regressions on three health-related outcomes, potentially yielding some insights into the underlying mechanism that explains the differences in outcomes between males and females. Column (1) presents the point estimate from a regression using the standard model from equation (1) on the full sample with the proportion of males in each cell as the dependent variable. The coefficient suggests that the proportion of males is 7.2 percentage points lower in the exposed cohort. Columns (2) & (4) present the results of a regression in which the dependent variable is the average month of birth for children born between January and July in each year, for males and females separately. While the coefficient reveals that the exposed males were born on average 1 week earlier (0.24 months), the experiment does not seem to have had any similar effect on the average birth month of females. Similarly, the cohort of men born in the wake of the experiment is significantly smaller, while no such effect is recognized for females (columns 3 and 5).

These results are in line with a large body of medical and biological studies suggesting that male are more sensitive to adverse conditions in early life than females (see e.g. Lee et al. 1998; Wells, 2000). Moreover, these estimates are consistent with results first found by Little et al. (1986) who, after controlling for a number of maternal background characteristics, found “a greater vulnerability of the male to alcohol exposure in the late first and early second trimester...” as measured by birth weight.³⁴

The instigators of the experiment suggested that at least some of the increased sales of strong beer were due to cross-border shopping by individuals from neighboring counties. In *Table 6* we examine to what extent such cross-border shopping also resulted in adverse outcomes for the children born in these counties. Remember that in the previous regressions these children were excluded from the sample. *Table 6* reports coefficients from the same specifications as in the tables above but now the “in utero” dummy is equal to 1 for the cohort of children born between April and July 1968 by mothers under the age of 21 in one of the five counties *neighboring* the experiment area.³⁵ The results from this exercise suggest that cross-border shopping did not affect the outcomes of the children in the neighbouring counties to any major extent. None of the coefficients is significantly different from zero at any conventional level of significance. Given that the neighbouring counties and the treatment counties are highly

³⁴ Using a sample of non-smoking, non-alcoholic women Little et al. related average daily consumption both in the week before pregnancy recognition (week 6 on average) and in the week prior to the first prenatal visit (between week 8 through 16, mean: 11.2) to birth weight. The differing effects between males and females on birth weight are particularly strong in the later case. Interestingly, the fraction of male births in their sample is also strongly negatively correlated with consumption during the same period of gestation. Furthermore, the results are consistent with differences in sensitivity to binge alcohol exposure displayed among male and female rats found by Goodlett and Peterson (1995).

³⁵ We exclude the experiment-region children from these regressions.

interdependent today and constitute a local labor market, this exercise also strengthens the case for our estimation strategy.

Table 7 examines the impact of the experiment on those who were between 1 to 12 months (panel A), and 13 to 24 months old (panel B) at the start of the experiment. Besides including dummies for the new cohorts of interest we also include the original “in utero” dummy to see to what extent the baseline results are sensitive to the change in specification. Interestingly the experiment does not seem to have had an effect on the outcomes of children born just prior to its implementation. We interpret this finding as evidence that it is in fact prenatal exposure to alcohol rather than an increased incidence of detrimental postnatal events caused by the experiment that drives the main results. Moreover, the respecified model yields qualitatively similar results as the baseline model which is reassuring.

Table 8 reports the impact of the experiment on children of mothers in late pregnancy (months 5-9) at the start of the experiment vs. the original treatment cohort. Only the probability of having graduated from high school seems to have been significantly affected in the earlier cohort, whereas the estimates of the impact on the original cohort are virtually identical with the main results. One might be tempted to interpret the results of this exercise as evidence that alcohol exposure during the first and second trimester is more detrimental than exposure later on. However, these findings could also merely reflect heterogeneous consumption responses to the increase in alcohol availability between mothers in early and late pregnancy. Unfortunately, the estimation strategy we employ here does not allow us to distinguish between these two mechanisms.

In order to attain a clearer picture of the dynamics of the impact of the experiment, *Table 9* reports estimates from regressions using monthly rather than quarterly data. Specifically we now let the treatment window slide over the cohorts potentially affected by the experiment. Hence, rather than just looking at those with the maximum amount of in utero exposure to the experiment, we now start with those born between November 1967 and February 1968, continuing with December 1967 through March 1968, up to those born between September 1968 and December 1968. The treatment window used in the main analysis, April through July 1968, is highlighted in bold. The treatment windows to the left of the vertical dashed line (columns I-VI) only contain cohorts estimated to have been conceived before the experiment started. To the right of the dashed line, at least some of the children in the treated cohorts were conceived during the course of the experiment. The parameter estimates reported follow a clear pattern. While there are no significant differences for the children with the least amount of exposure (reported in column I and II), there is an increasingly negative trend in outcomes as the treatment window is rolled towards the most exposed cohorts. For the educational outcomes, the strongest negative effect is reached somewhere between March and August 1968 (columns V - VII), as is the case for earnings.

In the case of years of education and earnings, we have performed the same analysis for each cohort born from three years before the main cohort until three years after. The parameter estimates from these regressions is summarized in *Figure 7*. The estimates reported between the two vertical dashed lines contain at least 1 cohort exposed to the experiment. Firstly, from this figure we can see more clearly that the timing in the dip in relative outcomes among the highest exposed cohorts is unusually large and fits very

well with the number of weeks of exposure. Secondly, while there are also dips for other cohorts for each one of the outcomes, during the experiment the change in both educational outcomes and earnings follows each other very well. Thirdly, interestingly in the case of education the estimates suggest that the children conceived at the end of the experiment period (i.e. born in the spring of 1969) have a relatively *higher* level of educational attainments ($p < 0.05$). This effect could in part be due to a *positive* effect of the experiment on parental composition. From previous research we know that alcohol consumption increases risky behaviour among young people.³⁶ Hence, if the higher alcohol consumption increased fertility relatively more among high ability parents this may explain the relative increase in educational attainments among the cohort conceived at the end of the experiment period.³⁷

To be able to test this hypothesis directly, we would ideally like to have some parental quality indicator measured *prior* to birth of the child. As such a measure is not available to us, we look at whether the fraction of children born by a mother with a high school degree (measured in 1990) is higher among those conceived during last part of the experiment.³⁸ This exercise indeed indicates that parental composition improved significantly for those children conceived during the later part of the experiment as the fraction of children born by mothers who graduated from high school increased by 3.3

³⁶ See e.g. Carpenter (2005), Grossman and Markowitz (2005).

³⁷ In the absence of legalized abortions (not freely available until 1975), there are several potential reasons for such effects to occur. One reason is that highly skilled women are assumingly less likely to become pregnant at an early age, as the cost of having a child is higher in terms of lost future earnings relative to low skill women. Hence, increased alcohol availability may have a larger *relative* affect on the pregnancy rate among highly skilled women than low-skilled women.

³⁸ Note that for these children conception was potentially affected by the experiment, although the time in utero during the experiment was short.

percentage points (mean=0.13 , $p<0.05$).³⁹ An additional finding that indirectly supports the idea that the relative increase in educational attainments are caused by the experiment is that the positive effect on education dies out directly after the last “treated” cohort leaves the treatment window (the cohorts just after the right vertical dashed lined in *Figure 7*).

The pattern in *Figure 7* also suggests that in order to identify the effects of a given alcohol policy intervention on children’s outcomes, it seems crucial to investigate whether parental composition and fertility rates have been affected too. Neglecting such effects may otherwise underestimate the true effect of the policy. However, for the cohorts for whom we in the present case can rule out direct effects on conception rates (i.e. for those conceived before the experiment started), increased alcohol exposure does indeed seem to have significant and economically important effects on adult outcomes.

5 Summary and conclusions

We employ a Swedish alcohol policy experiment conducted in the late 1960s to identify the impact of prenatal exposure to alcohol on adult labor market and educational outcomes. Young people under the age of 21 experienced the largest increase in alcohol availability during the experiment, and according to reports increased their alcohol consumption most. In line with these reports we find that the cohort of children born by mothers under the age of 21 and exposed to the experiment while in utero has

³⁹ This effect is driven by a 35 percent increase (est. 0.35, std.err 0.15) in the number of children born by a high school educated mother rather than a decrease in the number children born by a less educated mother. The estimates are attained by running the baseline regression with the fraction of mothers with a high school diploma as the dependent variable. We use the last cohort in which all children were conceived during the experiment (children born between January and April 1969) as the treatment group and also include a dummy for children born in the same

significantly reduced earnings, higher welfare dependence rates, and lower educational attainments compared with the surrounding cohorts. While we can not fully rule out that other unobserved factors correlated with alcohol consumption may explain at least part of the relatively large economic effects of prenatal alcohol exposure found here (e.g. smoking and illicit drug use), the magnitude and timing of the effects suggest that it is indeed alcohol that drives the results.⁴⁰

This paper is the first to address this issue within economics. It is also the first paper to apply a quasi-experimental strategy to identify the effect of maternal alcohol consumption during pregnancy on the child's outcomes. The findings in this paper add to a growing body of research documenting the effects of prenatal insults on adult outcomes. Together these studies suggest that investments in prenatal health may have long-term consequences not only for the individual but also generate multiplier effects that are beneficial to society as a whole.

Our study also provides evidence of an overlooked and potentially important mechanism that explains the linkage between teenage childbearing and child outcomes.⁴¹ Given the findings in this paper, identifying effective policy tools to reduce binge drinking among young people may not only improve the health of the individual, but also the outcomes of children born by teenage mothers.^{42 43}

months of 1968 in the specification. For the cohort size outcomes separate regressions are run for children born by a mothers with high school education and non-high school education.

⁴⁰ Attempts to assess the effects of alcohol use in comparison with the use of other drugs suggest that prenatal alcohol exposure may have broader and more long lasting effects compared to other drugs, see e.g. Day and Richardson (1994).

⁴¹ See e.g. Levine et al. (2001), Francesconi (2007), Hunt (2006) for evidence on the effect of teenage childbearing on offspring outcomes.

⁴² Tsai et al. (2007) use survey data to estimate the prevalence of binge drinking among women of child bearing age (18-44) in the US. In 2003 an estimated 7.2 million women (13 %) in these age categories engaged in binge drinking. In the early 1990s it was about 10 %. Among youths the binge drinking levels are even higher. About 90 % of the alcohol consumed by youths under the age of 21 in the United States is in the form of binge drinks (OJJDP, 2001).

A final caveat is in order before generalizing our results to other settings. It is not clear to what extent the results found here would emerge 30 years from now if a similar experiment was implemented is uncertain. For example, awareness of the health hazards of maternal drinking during pregnancy is arguably much greater now than it was 30 to 40 years ago. In some developing countries and among sub-populations in more developed countries reduced alcohol availability and/or increased information may however still prove to be an important tool for improving health and the economic outcomes of future generations.

While binge drinking decreased among youths up until the mid 1990s there are now signs of a reverse in this trend (Serdula et al., 2004).

⁴³ Carpenter et al. (2007) use data from 1976 through 2003 to estimate the impact of a variety of policy measures such as minimum legal drinking age laws, “zero tolerance” under age drunk driving laws and beer taxes on alcohol use among youths. They find that MLDA seems to have had significantly reduced alcohol consumption among high school seniors.

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Appendix A

Table A 1 Estimated prenatal exposure to the experiment

Month of birth	Est. date of conception [†]	Est. gestational age at start of experiment (month)	Minimum/Maximum number of weeks in utero during experiment		Trimester under exposure:	Experiment may have affected conception rate?
Before November 1967	Before February 1967	born	0	0	-	NO
November 1967	February 1967	8-9	0	4	3rd	NO
December 1967	March 1967	7-8	4	8	3rd	NO
January 1968	April 1967	6-7	8	12	3rd	NO
February 1968	May 1967	5-6	12	16	2nd, 3rd	NO
March 1968	June 1967	4-5	16	20	2nd, 3rd	NO
April 1968	July 1967	3-4	20	24	2nd, 3rd	NO
May 1968	August 1967	2-3	24	28	1st, 2nd, 3rd	NO
June 1968	September 1967	1-2	28	32	1st, 2nd, 3rd	NO
July 1968	October 1967	0-1	32	34	1st, 2nd, 3rd	NO
August 1968	November 1967	-	30	34	1st, 2nd, 3rd	YES
September 1968	December 1967	-	26	30	1st, 2nd, 3rd	YES
October 1968	January 1968	-	22	26	1st, 2nd, 3rd	YES
November 1968	February 1968	-	18	22	1st, 2nd	YES
December 1968	March 1968	-	14	18	1st, 2nd	YES
January 1969	April 1968	-	10	14	1st, 2nd	YES
February 1969	May 1968	-	6	10	1st	YES
March 1969	June 1968	-	2	6	1st	YES
April 1969	July 1968	-	0	2	1st	YES
After April 1969	After July 1968	-	0	0	-	NO

[†]These estimates all assume that conception occurred 9 months prior to birth. Experiment started on November 1st 1967 and ended on July 14th 1968. The cohorts highlighted in bold are those defined as treated in the main analysis.

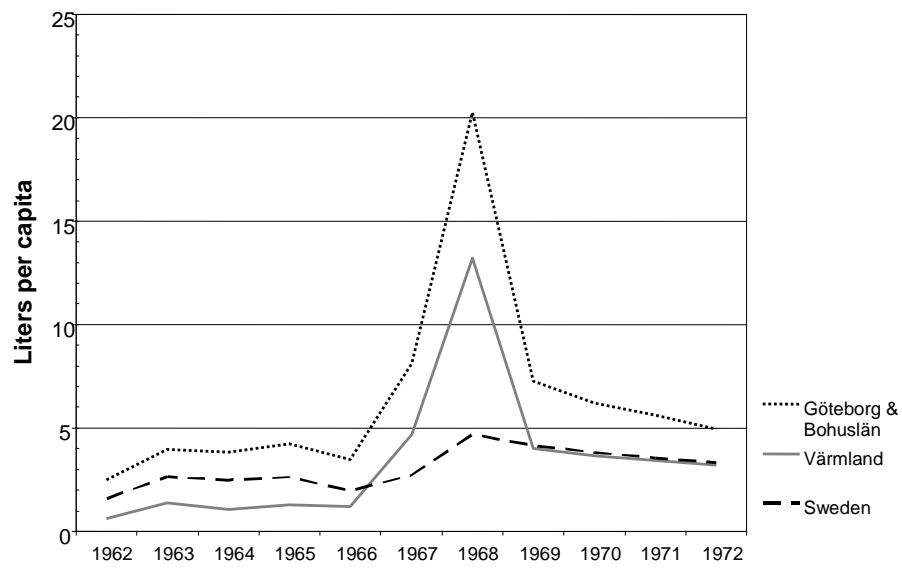


Figure 1 Yearly strong beer consumption per capita.

Source: SCB 1962-72

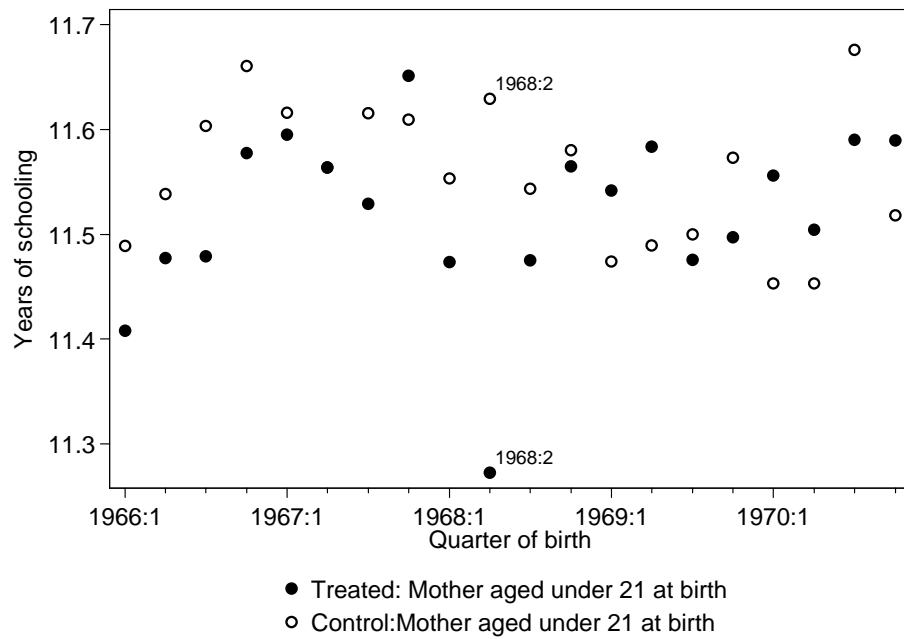


Figure 2 Average years of schooling, treated vs. control.

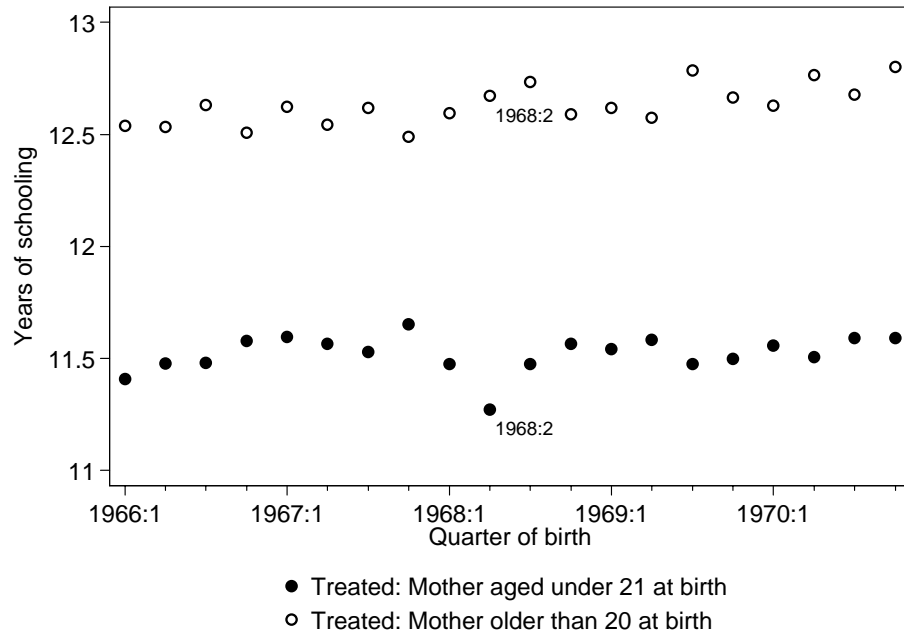


Figure 3 Average years of schooling, young vs. old mother.

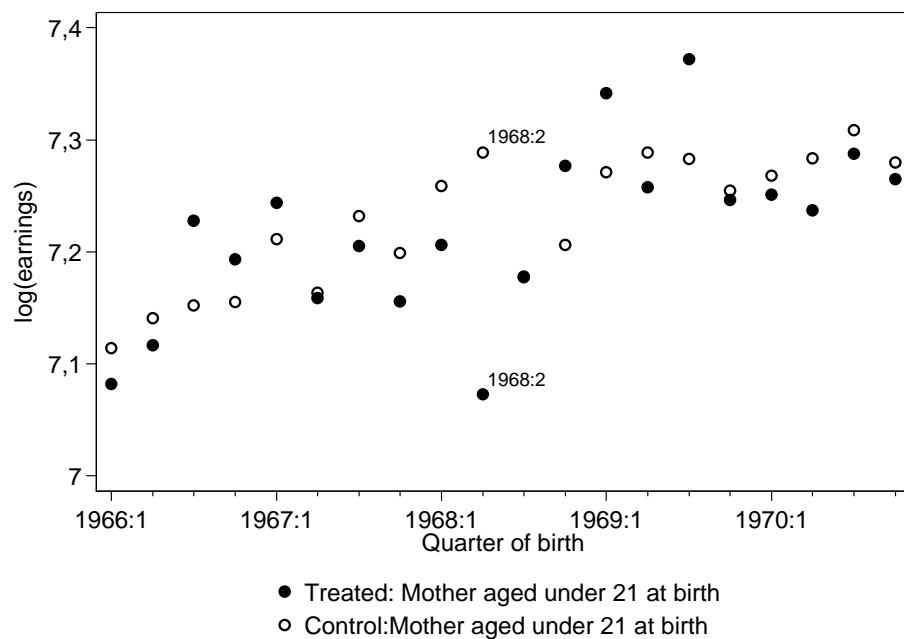


Figure 4 Average $\ln(\text{earnings})$ at age 32, treated vs. control.

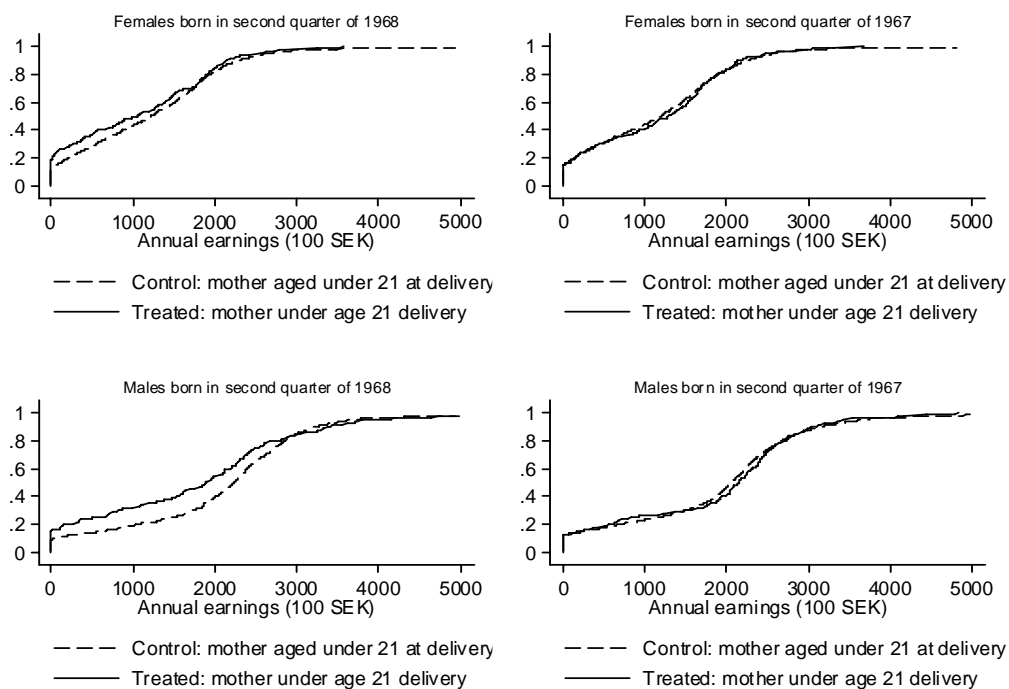


Figure 5 Cumulative distribution of earnings at age 32.

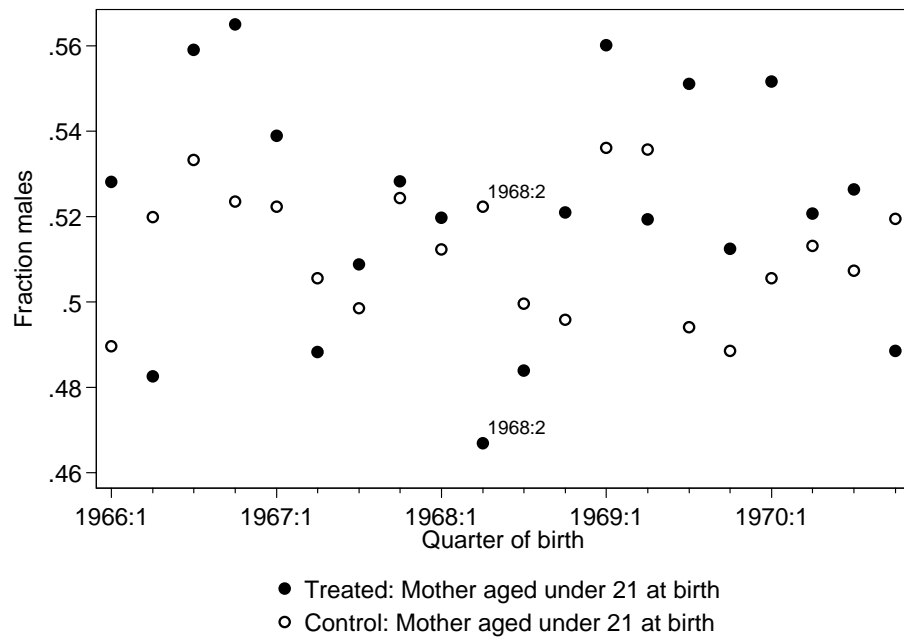


Figure 6 Sex-ratio in 2000

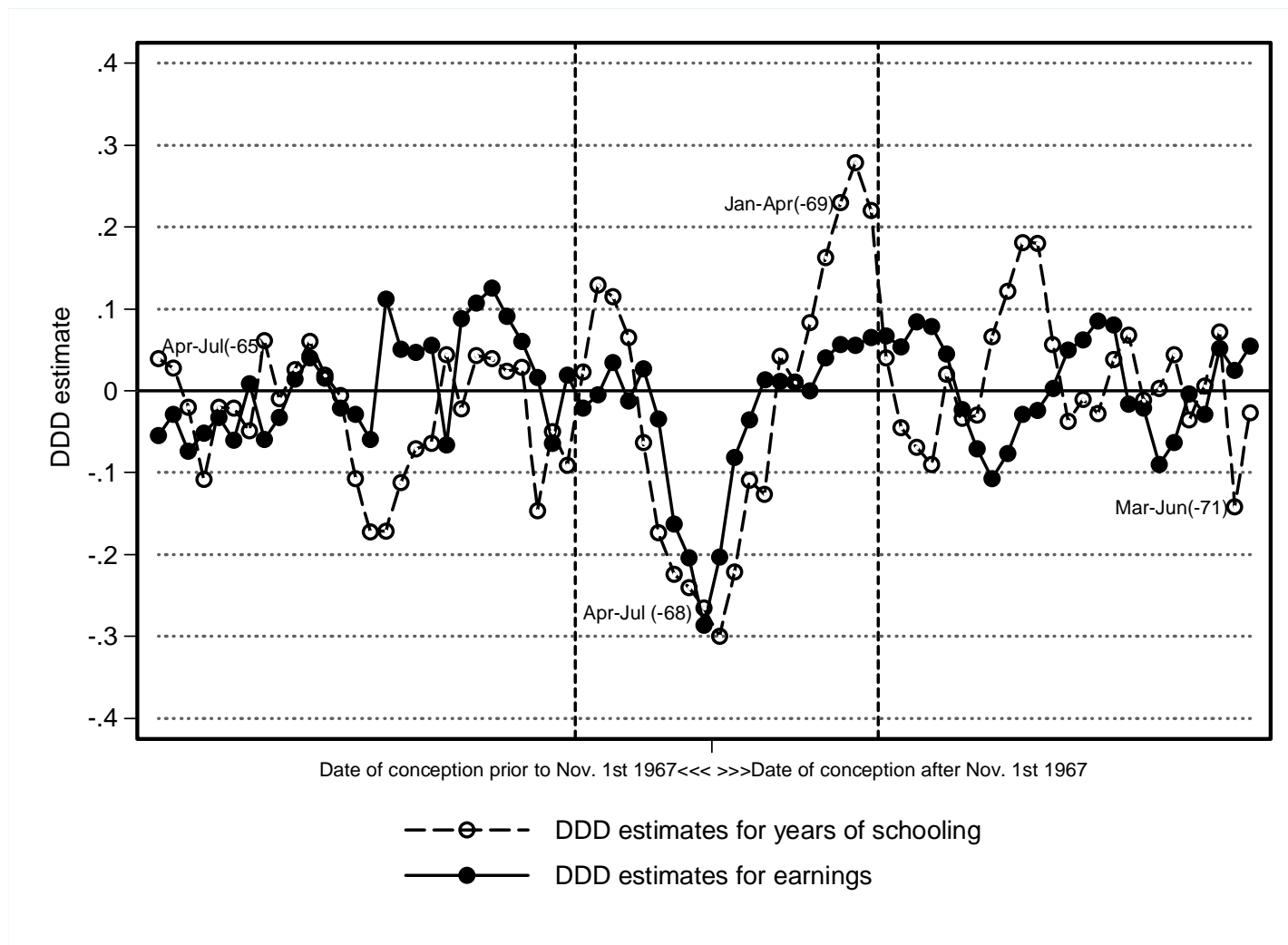


Figure 7 Difference-in-difference-in-differences estimates for years of schooling and earnings

Table 1 Means of background characteristics and outcomes (first two quarters of each year)

	TREATED			CONTROL			TREATED			CONTROL		
	ALL MOTHERS (I)			ALL MOTHERS (II)			YOUNG MOTHERS (III)			YOUNG MOTHERS (IV)		
	Born <1968	Born 1968	Born >1968	Born <1968	Born 1968	Born >1968	Born <1968	Born 1968	Born >1968	Born <1968	Born 1968	Born >1968
Outcomes:												
Education (years)	12.28	12.36	12.52	12.26	12.35	12.50	11.48	11.40	11.55	11.52	11.59	11.49
Fraction high school graduates	0.917	0.927	0.927	0.915	0.930	0.928	0.867	0.853	0.860	0.861	0.882	0.857
Fraction college graduates	0.162	0.163	0.173	0.160	0.156	0.174	0.064	0.052	0.064	0.068	0.068	0.064
Average log (yearly earnings) at age 32	7.206	7.341	7.402	7.213	7.333	7.406	7.091	7.143	7.302	7.1378	7.254	7.287
Percent w. zero earnings (age 32)	0.115	0.095	0.100	0.109	0.093	0.093	0.142	0.143	0.127	0.129	0.098	0.118
Percent on welfare in 2000	0.047	0.037	0.053	0.040	0.038	0.044	0.085	0.081	0.094	0.064	0.062	0.088
Fraction males	0.510	0.516	0.513	0.512	0.515	0.520	0.498	0.491	0.529	0.513	0.517	0.522
Family characteristics:												
Age of father at delivery	27.1	26.9	27.1	26.9	26.8	27.2	22.5	22.4	22.8	22.6	22.4	22.8
Age of mother at delivery	23.9	24.1	24.4	23.7	24.0	24.4	18.9	18.9	18.9	19.2	18.9	18.9
Mothers education (high school)	0.216	0.236	0.290	0.216	0.235	0.288	0.107	0.106	0.115	0.129	0.113	0.104
Average number of children in cells	464	443	430	310	300	279	238	197	158	168	126	103

Note: The table reports weighted averages over cells. There are 1, 748 county-by-quarter-by-young/old mother-cells.

Table 2 The impact of the experiment on educational attainments

A. DEPENDENT VARIABLE: Years of schooling	Sample		
	ALL (1)	MEN (2)	WOMEN (3)
In utero (month 1-4)	−0.266*** (0.049)	−0.473*** (0.124)	−0.101 (0.151)
Quarter of birth dummies	YES	YES	YES
County of birth dummies	YES	YES	YES
Mother under age 21 dummy	YES	YES	YES
Observations	1393	1388	1387
R-squared	0.98	0.96	0.95
Mean	12.33	12.18	12.49
B. DEPENDENT VARIABLE: Fraction high school graduates	Sample		
	ALL (1)	MEN (2)	WOMEN (3)
In utero (month 1-4)	−0.039*** (0.009)	−0.092*** (0.017)	0.015 (0.014)
Quarter of birth dummies	YES	YES	YES
County of birth dummies	YES	YES	YES
Mother under age 21 dummy	YES	YES	YES
Observations	1393	1388	1387
R-squared	0.90	0.85	0.82
Mean	0.921	0.910	0.934
C. DEPENDENT VARIABLE: Fraction graduated from higher education	Sample		
	ALL (1)	MEN (2)	WOMEN (3)
In utero (month 1-4)	−0.025** (0.012)	−0.039*** (0.013)	−0.021 (0.014)
Quarter of birth dummies	YES	YES	YES
County of birth dummies	YES	YES	YES
Mother under age 21 dummy	YES	YES	YES
Observations	1393	1388	1387
R-squared	0.95	0.92	0.92
Mean	0.159	0.138	0.181

Note: Each column and panel represents a separate regression. The dependent variable is years of schooling, fraction with higher education or fraction who have completed high school. The unit of observation is all first born children alive in 2000 either by mothers aged ≥ 21 or below in a given year, quarter and county. "In utero(month 1-4) " is a dummy equal to 1 if the child was born by a mother under age 21 and exposed to the experiment while *in utero* from early until late pregnancy (see section 3.1 for details). All regressions include year of birth, quarter of birth, county of birth, mother under age 21 at delivery dummies and a set of interaction terms between these variables (see equation 1). All regressions are weighted by the inverse of the cell size used to calculate the dependent variable. Heteroscedasticity robust standard errors are reported in parenthesis.

Table 3 The impact of the experiment on labor market outcomes

A.DEPENDENT VARIABLE: ln(earnings)	Sample		
	ALL (1)	MEN (2)	WOMEN (3)
In utero (month 1-4)	-0.241*** (0.053)	-0.228*** (0.081)	-0.177** (0.097)
Quarter of birth dummies	YES	YES	YES
County of birth dummies	YES	YES	YES
Mother under age 21 dummy	YES	YES	YES
Observations	1391	1387	1388
R-squared	0.88	0.87	0.79
Mean	7.26	7.57	6.93
B.DEPENDENT VARIABLE: Fraction with zero earnings	Sample		
	ALL (1)	MEN (2)	WOMEN (3)
In utero (month 1-4)	0.071*** (0.012)	0.069*** (0.017)	0.069*** (0.013)
Quarter of birth dummies	YES	YES	YES
County of birth dummies	YES	YES	YES
Mother under age 21 dummy	YES	YES	YES
Observations	1393	1388	1387
R-squared	0.76	0.71	0.67
Mean	0.10	0.09	0.11
C.DEPENDENT VARIABLE: Fraction welfare participants	Sample		
	ALL (1)	MEN (2)	WOMEN (3)
In utero (month 1-4)	0.036*** (0.009)	0.051*** (0.016)	0.021 (0.021)
Quarter of birth dummies	YES	YES	YES
County of birth dummies	YES	YES	YES
Mother under age 21 dummy	YES	YES	YES
Observations	1386	1386	1386
R-squared	0.84	0.74	0.76
Mean	0.042	0.039	0.046

Note: Each column and panel represents a separate regression. The dependent variable is average log earnings, fraction with zero income or fraction on welfare. The unit of observation in each regression is all children alive in 2000 born either by mothers aged ≥ 21 or below in a given year, quarter and county. "In utero(month 1-4)" is a dummy equal to 1 if the child was born by a mother under age 21 and exposed to the experiment while *in utero* from early until late pregnancy (see section 3.1 for details). All regressions include year of birth, quarter of birth, county of birth, mother under age 21 at delivery dummies and the corresponding interaction terms. Earning outcomes are measured at age 32 while fraction welfare recipients are measured in 2000. All regressions are weighted by the inverse of the cell size used to calculate the dependent variable. The earnings coefficient presented is given by the transformation $(\exp(\beta)-1)$. Heteroscedasticity robust standard errors are reported in parenthesis.

Table 4 The impact of the experiment by maternal education and earnings: labor market and educational outcomes

DEPENDENT VARIABLES:												
A.	Years of schooling		High school graduates		Higher education		Earnings		Zero earnings		Welfare	
	High school grad	Not high school grad	High school grad	Not high school grad	High school grad	Not high school grad	High school grad	Not high school grad	High school grad	Not high school grad	High school grad	Not high school grad
Education of mother (1990):												
In utero (month 1-4)	-0.347* (0.182)	-0.200*** (0.063)	-0.020 (0.044)	-0.038*** (0.010)	-0.016 (0.090)	-0.027** (0.010)	-0.218 (0.185)	-0.235*** (0.046)	0.010 (0.064)	0.078*** (0.008)	0.058 (0.038)	0.025** (0.013)
# observations	1350	1350	1350	1350	1350	1350	1350	1350	1350	1350	1350	1350
B.	Years of schooling		High school graduates		Higher education		Earnings		Zero earnings		Welfare	
	Above median	Below median	Above median	Below median	Above median	Below median	Above median	Below median	Above median	Below median	Above median	Below median
Mothers labor earnings (1990):												
In utero (month 1-4)	-0.071 0.103	-0.360*** (0.083)	-0.028 (0.028)	-0.045* (0.025)	-0.022 (0.027)	-0.021* (0.011)	-0.226 (0.216)	-0.248 (0.187)	0.042*** (0.015)	0.092*** (0.020)	0.017* (0.009)	0.047*** (0.014)
# observations	1350	1350	1350	1350	1350	1350	1350	1350	1350	1350	1350	1350

Note: Each reported column represents a separate regression. The outcomes are measured within each county of birth/year of birth/quarter of birth/mom<age 21 at delivery cell. All regressions are weighted by the inverse of the cell size used to calculate the dependent variable. Heteroscedasticity robust standard errors are reported in parenthesis.

Table 5 The impact of the experiment on health related outcomes

	DEPENDENT VARIABLES:				
	Fraction	Month of birth	ln	Month of birth	ln
	males		(cohort size)		(cohort size)
	(1)	(2)	(3)	(4)	(5)
	ALL	MEN	MEN	WOMEN	WOMEN
In utero	−0.072*** (0.024)	0.240** (0.122)	−0.166*** (0.055)	0.042 (0.146)	0.134* (0.070)
Year/Quarter dummies	YES	YES(YEAR)	YES	YES(YEAR)	YES
C.O.B dummies	YES	YES	YES	YES	YES
Mom age<21 dummy	YES	YES	YES	YES	YES
Observations	1393	359	1385	354	1386
R-squared	0.56	0.65	0.98	0.63	0.98
Mean(not logs)	0.515	4.00	124.97	4.00	117.85

Note: Each column and panel represents a separate regression. Except for when the dependent variable is “month of birth” the outcomes are measured within each county of birth/year of birth/quarter of birth/mom<age 21 at delivery cell. In the “month of birth” case instead the analysis each cell refers to county/year of birth/mother under age 21 cell averages. Furthermore, in this case only those born between January through July is retained. “*In utero*” is a dummy equal to 1 if the child was born by a mother under age 21 and exposed to the experiment while *in utero* (see text for details). All regressions include year of birth, quarter of birth, county of birth, mother under age 21 at delivery dummies and the corresponding interaction terms. All regressions are weighted by the inverse of the cell size used to calculate the dependent variable, except for the cohort size outcome. Heteroscedasticity robust standard errors are reported in parenthesis.

Table 6 The impact of the experiment on neighboring counties: labor market, educational and health outcomes

DEPENDENT VARIABLES: Labor and education												
A.	Years of schooling		High school graduates		Higher education		Earnings		Zero earnings		Welfare	
Sample	Men	Women	Men	Women	Men	Women	Men	Women	Men	Women	Men	Women
In utero (month 1-4)	- 0.106 (0.135)	- 0.140 (0.088)	0.006 (0.027)	- 0.026 (0.022)	- 0.021 (0.014)	0.007 (0.023)	0.040 (0.082)	0.101 (0.092)	0.016 (0.019)	0.029 (0.018)	0.003 (0.019)	0.017 (0.016)
Number of observations	1598	1598	1598	1598	1598	1598	1598	1598	1598	1598	1598	1598
DEPENDENT VARIABLES: Health												
B.	Fraction males		Month of birth		ln(cohort size)							
Sample	ALL		Men	Women	Men	Women						
In utero	- 0.006 (0.025)		0.119 (0.085)	- 0.037 (0.123)	0.022 (0.097)	0.037 (0.074)						
Number of observations	1598		413	408	1598	1598						

Note: Each column and panel represents a separate regression. Except for when the dependent variable is “month of birth” the outcomes are measured within each county of birth/year of birth/quarter of birth/mom<age 21 at delivery cell. In the “month of birth” case instead the analysis each cell refers to county/year of birth/mother under age 21 cell averages. Furthermore, in this case only those born between January and July are retained. All regressions are weighted by the inverse of the cell size used to calculate the dependent variable, except for the cohort size outcome case. Heteroscedasticity robust standard errors are reported in parenthesis.

Table 7 The impact of the experiment on children aged 1-12 months and 13-24 months at the start of the experiment: Labor market and educational outcomes

DEPENDENT VARIABLES:						
A.	Years of schooling	High school graduates	Higher education	Earnings	Zero earnings	Welfare
AGE AT START OF EXPERIMENT:	ALL	ALL	ALL	ALL	ALL	ALL
I(1-12 months)	-0.034 (0.045)	-0.0003 (0.009)	0.0004 (0.010)	0.041 (0.034)	0.0004 (0.010)	-0.006 (0.011)
In utero (month 1-4)	-0.271*** (0.050)	-0.039*** (0.009)	-0.025** (0.012)	-0.240*** (0.053)	0.071*** (0.0122)	0.035*** (0.009)
Number of observations	1394	1394	1394	1394	1394	1394
DEPENDENT VARIABLES:						
B.	Years of schooling	High school graduates	Higher education	Earnings	Zero earnings	Welfare
AGE AT START OF EXPERIMENT:	ALL	ALL	ALL	ALL	ALL	ALL
I(13-24 months)	-0.056 (0.071)	-0.002 (0.016)	-0.007 (0.009)	-0.014 (0.030)	0.002 0.012	0.002 (0.006)
In utero (month 1-4)	-0.263*** (0.053)	-0.039*** (0.009)	-0.024** (0.012)	-0.240*** (0.046)	0.071*** (0.012)	0.036*** (0.009)
Number of observations	1394	1394	1394	1394	1394	1394

Note: Each column and panel (A & B) represents a separate regression. The outcomes are measured within each county of birth/year of birth/quarter of birth/mom<age 21 at delivery cell. Robust standard errors in parenthesis. The *I(1-12)* take the value 1 if the child was born in 1966Q4-1967Q3 and zero otherwise. The "*In utero*" dummy is equal to 1 if the child was born between April and July 1968. All regressions are weighted by the inverse of the cell size used to calculate the dependent variable. Heteroscedasticity robust standard errors are reported in parenthesis.

Table 8 The impact of the experiment on children of mothers in depending on gestational age.
Late pregnancy (month 5-9) vs. early pregnancy (month 1-4) at start of experiment: Labor market and educational outcomes

	DEPENDENT VARIABLES: Labor and education					
	Years of schooling	High school graduates	Higher education	Earnings	Zero earnings	Welfare
Gestational age at start of experiment:	ALL	ALL	ALL	ALL	ALL	ALL
In utero (month 5-9)	0.036 (0.097)	-0.019** (0.009)	0.014 (0.021)	0.032 (0.075)	-0.005 (0.016)	0.004 (0.006)
In utero (month 1-4)	-0.256*** (0.063)	-0.043*** (0.010)	-0.023* (0.013)	-0.242*** (0.053)	0.070*** (0.013)	0.033*** (0.010)
Number of observations	1394	1394	1394	1394	1394	1394

Note: Each column represents a separate regression. The outcomes are measured within each county of birth/year of birth/quarter of birth/mom<age 21 at delivery cell. "In utero (month 5-9)" is equal to 1 if the child the child was born between November 1967 and March 1968. "In utero (month 1-4)" refers as above to the original treatment cohort, those born between April and July 1968. All regressions are weighted by the inverse of the cell size used to calculate the dependent variable. Heteroscedasticity robust standard errors are reported in parenthesis.

Table 9 The impact of the experiment on children depending on gestational age at start of experiment (monthly data)

DEPENDENT VARIABLES: Educational, labor market and health-related outcomes											
	(I)	(II)	(III)	(IV)	(V)	(VI)	(VII)	(VIII)	(IX)	(X)	(XI)
Period of Birth	Nov-Feb	Dec-Mar	Jan-Apr	Feb-May	Mar-Jun	Apr-Jul	May-Aug	Jun-Sept	Jul-Oct	Aug-Nov	Sept-Dec
Est. gestational age (months) in November 1967	(6-9)	(5-8)	(4-7)	(3-6)	(2-5)	(1-4)	(n.c.-3)	(n.c.-2)	(n.c.-1)	No one conceived	No one conceived
<i>Outcome:</i>											
Years of Schooling	0.065 (0.134)	-0.063 (0.079)	-0.173** (0.074)	-0.224*** (0.075)	-0.240*** (0.082)	-0.266*** (0.083)	-0.300*** (0.95)	-0.220** (0.112)	-0.110 (0.108)	-0.130 (0.101)	0.043 (0.097)
High School grad.	-0.002 (0.015)	-0.014 (0.017)	-0.030* (0.016)	-0.026* (0.015)	-0.044*** (0.012)	-0.037** (0.016)	-0.036* (0.020)	-0.019 (0.022)	-0.013 (0.019)	-0.009 (0.017)	0.007 (0.016)
University grad.	0.012 (0.025)	-0.011 (0.017)	-0.017 (0.016)	-0.018 (0.015)	-0.015 (0.016)	-0.023** (0.012)	-0.036*** (0.013)	-0.030* (0.018)	-0.010 (0.020)	-0.017 (0.020)	0.001 (0.020)
Labor earnings	-0.012 (0.043)	0.026 (0.086)	-0.035 (0.102)	-0.163 (0.119)	-0.204* (0.118)	-0.290*** (0.092)	-0.203* (0.109)	-0.081 (0.068)	-0.040 (0.072)	0.014 (0.076)	0.011 (0.079)
Zero earnings	-0.008 (0.021)	0.016 (0.018)	0.051* (0.029)	0.071*** (0.024)	0.076*** (0.024)	0.072*** (0.026)	0.034** (0.017)	0.011 (0.023)	-0.008 (0.021)	-0.016 (0.018)	-0.036*** (0.013)
Welfare dependency	-0.001 (0.016)	0.005 (0.013)	0.017 (0.018)	0.012 (0.017)	0.022 (0.016)	0.034** (0.015)	0.017* (0.010)	0.013 (0.013)	0.007 (0.013)	0.003 (0.013)	0.002 (0.013)
Fraction males	-0.002 (0.028)	0.004 (0.024)	-0.008 (0.028)	-0.058** (0.027)	-0.064** (0.028)	-0.073** (0.029)	-0.039 (0.033)	-0.025 (0.031)	-0.015 (0.040)	-0.001 (0.041)	0.003 (0.043)
Number of observations	4240	4240	4240	4240	4240	4240	4240	4240	4240	4240	4240

Note: Each column and panel represents a separate regression using the model in equation (1). The outcomes are averages/fractions within each county of birth/month of birth/mom<age 21 at delivery cell. All regressions are weighted by the inverse of the cell size used to calculate the dependent variable. Heteroscedasticity robust standard errors are reported in parenthesis. The estimates from using the original treatment window are reported in bold (column VI).